

REPORT DOCUMENTATION PAGE

Form Approved
OMB No. 0704-0188

Public reporting burden for this collection of information is estimated to average 1 hour per response, including the time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing the collection of information. Send comments regarding this burden estimate or any other aspect of this collection of information, including suggestions for reducing this burden, to Washington Headquarters Services, Directorate for Information Operations and Reports, 1215 Jefferson Davis Highway, Suite 1204, Arlington, VA 22202-4302, and to the Office of Management and Budget, Paperwork Reduction Project (0704-0188), Washington, DC 20503.

1. AGENCY USE ONLY (Leave blank)		2. REPORT DATE January 1999		3. REPORT TYPE AND DATES COVERED Technical Report	
4. TITLE AND SUBTITLE Non-invasive method of inducing hypothermia in multiple rats simultaneously				5. FUNDING NUMBERS	
6. AUTHOR(S) Candace B. Matthew and Wiliam D. Schrock					
7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES) U.S. Army Research Institute of Environmental Medicine Natick, MA 01760-5007				8. PERFORMING ORGANIZATION REPORT NUMBER	
9. SPONSORING / MONITORING AGENCY NAME(S) AND ADDRESS(ES) Same as 7 Above				10. SPONSORING / MONITORING AGENCY REPORT NUMBER	
11. SUPPLEMENTARY NOTES					
12a. DISTRIBUTION / AVAILABILITY STATEMENT Unlimited				12b. DISTRIBUTION CODE	
13. ABSTRACT (Maximum 200 words) <p>Hypothermia has been and continues to be a problem faced by military personnel when operating in cold environments or when water immersion in moderate temperatures results in the loss of body heat that is not compensated for by heat production. Vascular leakage of large protein molecules and accompanying water or extravasation manifested in a loss of vascular volume is one of the pathophysiological findings resulting from rewarming following hypothermia. Recent work in this laboratory with hyperthermic rats has shown that hyperthermia-induced extravasation can be quantitated and the organ sites located using recovery of Evans Blue from tissues. Recovery of fluorescent microspheres from the same rats indicated a change in reticuloendothelial system function with hyperthermia. Prior to using the same techniques to quantitate these changes following hypothermia and rewarming, it was necessary to develop a non-invasive method of inducing closely controlled hypothermia in rats. This report describes the construction of three water cooled copper coils into which anesthetized rats were placed. Valves in the parallel tubing connecting these coils allowed the cooling and subsequent rewarming of three rats simultaneously with only one circulating water bath. Data on 3 sets of 3 rats are presented.</p> <p>19990119 116</p>					
14. SUBJECT TERMS Hypothermia, animal model, rat, telemetry, rewarming				15. NUMBER OF PAGES	
				16. PRICE CODE	
17. SECURITY CLASSIFICATION OF REPORT Unclassified	18. SECURITY CLASSIFICATION OF THIS PAGE Unclassified	19. SECURITY CLASSIFICATION OF ABSTRACT Unclassified	20. LIMITATION OF ABSTRACT UL		

Approved for public release
distribution unlimited

TECHNICAL REPORT
NO. T

Non-invasive method of inducing hypothermia in multiple rats simultaneously

Candace B. Matthew and William D. Schrock

Project Reference: 30161102BS15

Military Nutrition and Biochemistry Division
U.S. Army Research Institute of Environmental Medicine
Natick, Massachusetts 01760-5007

CONTENTS

LIST OF FIGURES	iv
EXECUTIVE SUMMARY	v
INTRODUCTION	1
METHODS AND MATERIALS	3
RESULTS	4
DISCUSSION	5
ACKNOWLEDGMENT	5
REFERENCES	5
FIGURES	8
DISTRIBUTION LIST	13

LIST OF FIGURES

FIGURE	LEGEND	PAGE
1	Individual coil into which rats are placed for induction of hypothermia	8
2	Schematic of 3-rat cooling system	9
3	Cooling curves of 3 rats from 8 Oct 98	10
4	Cooling curves of 3 rats from 20 Oct 98 including coil temperature	11
5	Cooling curves of 3 rats from 23 Oct 98 indicating tighter control	12

EXECUTIVE SUMMARY

Hypothermia has been and continues to be a problem faced by military personnel when operating in cold environments or when water immersion in moderate temperatures results in the loss of body heat that is not compensated for by heat production. Vascular leakage of large protein molecules and accompanying water or extravasation manifested in a loss of vascular volume is one of the pathophysiological findings resulting from rewarming following hypothermia. Recent work in this laboratory with hyperthermic rats has shown that hyperthermia-induced extravasation can be quantitated and the organ sites located using recovery of Evans Blue from tissues. Recovery of fluorescent microspheres from the same rats indicated a change in reticuloendothelial system function with hyperthermia. Prior to using the same techniques to quantitate these changes following hypothermia and rewarming, it was necessary to develop a non-invasive method of inducing closely controlled hypothermia in rats. This report describes the construction of three water cooled copper coils into which anesthetized rats were placed. Valves in the parallel tubing connecting these coils allowed the cooling and subsequent rewarming of three rats simultaneously with only one circulating water bath. Data on 3 sets of 3 rats are presented.

INTRODUCTION

Hypothermia- Historically, cold ambient temperatures have resulted in substantial numbers of military casualties due to exposure to the elements resulting in hypothermia. Recent events have served to demonstrate that the problems of cold exposure still hamper soldier effectiveness. During February 1995, four Ranger students died of hypothermia while undergoing partial immersion in swamp training at Camp Rudder, Pensacola, Florida. British troops sustained numerous hypothermia-related injuries in the Falklands in 1992, and the current operating conditions of our soldiers in Bosnia expose them to the risks of hypothermia.

Accidental hypothermia is an acute drop in core temperature (T_{core}) usually in cold environments without any pathology of the thermoregulatory system. Hypothermia is more common in the elderly (reduced metabolic rate and insensitivity to cold) or the very young (increased heat loss due to a large surface area to mass ratio), but it is also seen in fit individuals working to exhaustion in cold environments (2,18,22). As body temperature falls there is a decrease in blood flow to the extremities to conserve heat in the heart and brain. Also, shivering is initiated in an attempt to increase metabolic heat production. In humans, mild hypothermia (T_{core} 34-36°C) is characterized by increases in heart rate, peripheral vascular resistance, blood pressure (BP), and cardiac output (CO). Moderate hypothermia (T_{core} 30-34°C) is characterized by decreases in heart rate, BP, and CO as well as the occasional appearance of arrhythmias. Severe hypothermia (T_{core} <30°C) is characterized by intensifying symptomatology and the appearance of the characteristic "J" or Osborn wave in the ECG (22). Patients with severe hypothermia may appear clinically dead with marked decrease in cerebral blood flow, CO and arterial pressure.

Clinical Uses of Hypothermia- While controlled mild to moderate hypothermia is commonly used during many surgical procedures to lower metabolic rate and reduce the dangers of ischemia during temporary diversion of the blood supply from target organs (1,8), deep hypothermia can have serious pathophysiological consequences. In a clinical setting moderate hypothermia has reduced the severity of damage from traumatic brain injury in humans (12) and mild hypothermia decreased spinal cord injury in rabbits (13).

Hypothermic Pathophysiology- Rewarming from accidental severe hypothermia is associated with sudden, often fatal, vascular collapse characterized by falling CO and sudden drops in peripheral resistance and BP (16,20,21). During severe hypothermia in rats there is a decrease in blood flow to brain, heart, liver, spleen, kidney, intestines, and muscle to 3-33% of normal values; even upon rewarming these flows may still be depressed to 5-55% of pre-hypothermic values (21). Cooled blood circulating through the brain disrupted the blood brain barrier (15) while hypothermia altered endothelial cell structure (8). Cold exposure results in loss of transmembrane ion gradients and barrier function. Ion pump failure results in increased Ca^{++} concentration and consequent cytoskeletal disruption followed by organ damage due to the breakdown of endothelial integrity (8). Exposure to cold air results in epithelial damage and vascular leakage in the respiratory system (7,24). An increase in the viscosity of the blood results in extravasation of plasma proteins and water (25). This loss of fluid from the vascular bed is postulated to be responsible for the diminished CO (1) which may result in terminal

vascular collapse. Work on an isolated cat limb model (23,25) indicates that there is extravasation in peripheral vasculature, but no attempt was made to evaluate extravasation in specific organs or to ameliorate the problem.

Animal models of hypothermia- Many methods have been described for cooling the rat (17): hypercapnic hypoxia followed by placement in a glass chamber in a cold water bath; maintenance in a 4°C environment following injection of chlorpromazine sc 20 mg/kg; immersion to the neck in an ice water bath; cooling after insulin injection by confinement in a water bath or cold room; starvation for 2 or more days before cooling; cooling with sodium pentobarbital (17). In addition to chlorpromazine (5,9,11,17) L-tryptophan (3,5), salicylate (4), tyrosine (4), and 5-thio-D-glucose (6) have all been used to induce hypothermia by drug administration combined with cold exposure.

In a study of auditory evoked responses, rats were made hypothermic (31.5°C) by administration of sodium pentobarbital followed by exposure to $T_{amb} = 4^{\circ}\text{C}$ until the desired T_{core} was achieved (10). Halothane anesthesia and a cold water blanket were used to induce hypothermia to examine effects on amino acid release following ischemia (14). In a more extreme study, rats were anesthetized with sodium pentobarbital and T_{core} reduced to 13°C with ventilatory support below 20°C by insertion of U-shaped PE tubing into the rectum and esophagus through which water circulated for cooling and rewarming (20,21).

For this study, it was necessary to develop a method of non-invasively inducing hypothermia in rats with minimal stress. Invasive methods, immersion studies, or drug-induced methods have potential for altering vascular permeability, reticuloendothelial system function, or the blood pressure, heart rate and ECG measurements planned for future studies.

MATERIALS AND METHODS

Animals- Adult male ($N = 9$, $490 \pm 27\text{g}$) Sprague-Dawley rats, (Harlan Sprague Dawley, Inc. Indianapolis, IN) were housed in the USARIEM animal colony until the start of the experiment. The rats were maintained at 26°C and 50% rh and were caged individually in wire bottomed cages. Lighting was controlled automatically (on, 0600-1800 h) and Purina rat chow and water were available *ad lib* except during the experimental interval. The ambient temperature in the experimental room was 20-23°C.

Cooling apparatus- The individual coils (Fig. 1) into which the rats were placed were constructed of ¼" O.D. copper refrigeration tubing. The tubing was bent by hand into a coil of 14 turns; the opening at the large end through which the rat enters is 3.5" and at the small end is 2", requiring approximately 7 ft of tubing for each coil. The coil thus formed may be easily expanded or contracted to accommodate different sized animals.

The coil is supported on a platform (Fig. 1) made of plexiglass and stainless steel rods; this is derived by removing rods from a previously described (19) restraining cage. The 3 coils are connected to a water bath in parallel (Fig. 2) using vinyl tubing (¼" I.D.), "Y" and "T"

connectors, and hose clamps. A stopcock was placed at the inlet of each coil to regulate flow to each coil individually. Surface thermistors were placed at the inlet of each coil.

Cooling procedure- Rats were lightly anesthetized (sodium pentobarbital, 35 mg/kg, ip); 15 min after anesthesia administration, a rectal probe (Tcore) was inserted 6 cm beyond the anal sphincter and taped in place, and then each animal was placed in a coil. The coil containing the rat was then lightly wrapped with "bubble wrap" to provide insulation that neither prevented observation of the animal nor interfered with respiration.

Hypothermia was induced by a ramped drop in the temperature of the water from the water bath. The bath temperature (Tbath) was started at 15°C and then the temperature setting dropped to 10, 5 and finally 3°C at 5 to 10 min intervals. The water bath lagged behind the setting so that the water entering the coil did not reach 5°C until 30 min into the cooling procedure. The coil temperature (Tcoil) was never lower than 5°C indicating that there was a 2°C rise in water temperature between the water bath and the coil. Rats were taken to a Tcore as low as 19°C and maintained there for 30 min when a staged increase in the Tbath was used to rewarm the animals.

RESULTS

Figures 3-5 each represent the cooling curves of 3 rats cooled simultaneously on 3 different days. At time 0 (Fig. 3), Tbath was 15°C; over the next 15 min. the Tbath set point was moved to 5°C, but the water entering the coils (Tcoil) did not stabilize at 7°C until 30 min. At 45 min the Tbath set point was lowered to 3°C and Tcoil stabilized at 5°C by 60 min. At 80 min. the Tcore of rat 98218 was dropping at a faster rate than that of the other 2 animals; therefore, the flow into its coil was stopped which decreased the rate of drop of Tcore. At 100 and 110 min. the flow was terminated for #'s 98217 and 98219 respectively. The lowest Tcore attained was 23.4°C for 98218 at 100 min. At 120 min. the Tbath was increased to 25°C and at 125 min the flow was resumed in all coils. At 130 min Tbath was increased to 30°C and at 150 min was changed to 35°C. By 180 min. all animals had regained a Tcore of at least 35°C and were fully recovered from anesthesia.

In Fig. 4, the Tcore of 3 additional animals and the Tcoil are illustrated. The Tbath was decreased from 15°C to 3°C over the first 20 min. with the water entering the coil stabilizing at 5°C at 30 min. Rat 98234 cooled faster than the others; at 40 min. the flow to its coil was stopped, the insulation removed, and Tcore was maintained at the same level for the next 30 min. When 98235's Tcore approached that of 98234, flow was resumed in 98234's coil and its Tcore then tracked that of the other animals. At 120 min., flow was stopped in all coils to maintain the Tcore's around 23°C for 30 min after which the Tbath was raised to 25°C, the flow restarted, and then Tbath ramped up to 35°C.

On the third trial day (Fig. 5), Tcore's of all animals were closely modulated by earlier control of flow. When Tcore of 98232 began to fall faster at 120 min, it was allowed to fall to 20°C, the lowest Tcore to be used in future work. Rewarming was as in Fig. 3 and 4.

DISCUSSION

Results presented demonstrate the uniform induction of hypothermia in 3 rats simultaneously. Figures 3-5 clearly indicate that it is possible to control finely each animal's Tcore drop to align rats by modulating flow through the coil and removal of the insulation. Thus, with a single water bath, 3 rats were cooled simultaneously and consistently. Carefully controlled hypothermia will be important to upcoming studies on the pathophysiological effects of deep hypothermia and rewarming.

In another pilot experiment, an animal equipped with a surgically implanted telemetry transmitter (Data Sciences) was anesthetized and placed in one of the coils. During the cooling of this animal (data not shown), the copper coil did not interfere with the telemetry signals. Therefore, this system will be used to obtain blood pressure, heart rate, and ECG in addition to body temperature during induction of hypothermia.

This method for inducing closely controlled rate, duration, and intensity of hypothermia will be used to quantitate hypothermic effects on extravasation and reticuloendothelial system function. Subsequently, this system will be ideal to evaluate potential pretreatment and treatment modalities for stabilizing the vasculature to prevent extravasation of critical volume and to aid in preventing the vascular collapse and potential mortality that may result on rewarming from hypothermia.

ACKNOWLEDGMENTS

The authors would like to thank Julie E. Matthew for the CAD drawing in Fig. 1 and Dr. Ralph P. Francesconi for reading the manuscript.

REFERENCES

- 1- Berne RM. Myocardial function in severe hypothermia. *Circ. Res.* 11:90-95, 1954.
- 2- Danzl DF, Pozos RS, and Hamlet MP. Accidental hypothermia. In: *Wilderness Medicine: Management of Wilderness and Environmental Emergencies.* Auerbach PS (ed), Mosby, St. Louis. 1995, p. 51-103.
- 3- Francesconi RP and Mager M. L-tryptophan: effects on body temperature in rats. *Am. J. Physiol.* 227:402-405, 1974.
- 4- Francesconi RP and Mager M. Salicylate, tryptophan, and tyrosine hypothermia. *Am. J. Physiol.* 228:1431-1435, 1975.
- 5- Francesconi RP and Mager M. Hypothermia induced by chlorpromazine or L-tryptophan: effects on treadmill performance in the heat. *J. Appl. Physiol.* 47:813-817, 1979.
- 6- Francesconi RP and Mager M. Hypothermia induced by 5-thio-D-glucose: Effects on treadmill performance in the heat. *Aviat. Space Environ. Med.* 51:754-758, 1980.
- 7- Giesbrecht GG. The respiratory system in a cold environment. *Aviat. Space Environ. Med.* 66:890-902, 1995.

- 8- Hansen TN, Dawson PE, and Brockbank KGM. Effects of hypothermia upon endothelial cells: mechanisms and clinical importance. *Cryobiology* 31:101-106, 1994.
- 9- Hultborn R, Lundgren-Eriksson L, Ottosson-Lönn S, Ryd W, and Weiss L. Chlorpromazine-induced hypothermia in tumor-bearing mice, acute cytotoxic drug lethality and long-term survival. *Acta Oncologica* 29:941-944, 1990.
- 10- Janssen R, Hetzler BE, Creason JP, and Dyer RS. Differential impact of hypothermia and pentobarbital on brain-stem auditory evoked responses. *Electroenceph. Clin. Neurophysiol.* 80:412-421, 1991.
- 11- Lundgren-Eriksson L, Carlsson A, Eksborg S, Ryd W, Vesanen R, and Hultborn R. Pharmacokinetics of doxorubicin and epirubicin in mice during chlorpromazine-induced hypothermia. *Cancer Chemother. Pharmacol.* 40:419-424, 1997.
- 12- Marion DW, Penrod LE, Kelsey SF, Obrist WD, Kochanek PM, Palmer AM, Wisniewski SR, and DeKosky ST. Treatment of traumatic brain injury with moderate hypothermia. *N. Engl. J. Med.* 336:540-546, 1997.
- 13- Matsumoto M, Iida Y, Sakabe T, Sano T, Ishikawa T, and Nakakimura K. Mild and moderate hypothermia provide better protection than a burst-suppression dose of thiopental against ischemic spinal cord injury in rabbits. *Anesthesiology* 86:1120-1127, 1997.
- 14- Nakashima K and Todd MM. Effects of hypothermia, pentobarbital, and isoflurane on postdepolarization amino acid release during complete global cerebral ischemia. *Anesthesiology* 85:161-168, 1996.
- 15- Öztaş B and Küçük. Intracarotid hypothermic saline infusion: a new method for reversible blood-brain barrier disruption in anesthetized rats. *Neurosci. Lett.* 190:203-206, 1995.
- 16- Popovic VP and Kent KM. Cardiovascular responses in prolonged hypothermia. *Am. J. Physiol.* 209:1069-1074, 1965.
- 17- Popovic V. Survival time of hypothermic white rats (15°C) and ground squirrels (10°C). *Am. J. Physiol.* 199:463-466, 1960.
- 18- Reuler JB. Hypothermia: Pathophysiology, clinical settings, and management. *Ann. Int. Med.* 89:519-527, 1978.
- 19- Thomas, G.J., C.B. Matthew, W.T. Matthew, and R.W. Hubbard. A system of rat restraint using a cone-shaped, plexiglass and stainless steel rod cage. USARIEM Tech Report No. T6/85.
- 20- Tveita T, Skandfer M, Refsum H, and Ytrehus K. Experimental hypothermia and rewarming: changes in mechanical function and metabolism of rat hearts. *J. Appl. Physiol.* 80:291-297, 1996.
- 21- Tveita T, Ytrehus K, Skandfer M, Øian P, Helset E, Myhre ESP, and Larsen TS. Changes in blood flow distribution and capillary function after deep hypothermia in rat. *Can. J. Physiol. Pharmacol.* 74:376-381, 1996.
- 22- Weinberg AD. Hypothermia. *Ann. Emerg. Med.* 22:370-377, 1993.
- 23- Wolf MB, Porter LP, Scott DRC and Zhang JX. Effects of cold on vascular permeability and edema formation in the isolated cat limb. *J. Appl Physiol.* 73:166-172, 1992.
- 24- Yoshihara S, Chan B, Yamawaki I, Geppetti P, Ricciardolo FLM, Massion PP, and Nadel JA. Plasma extravasation in the rat trachea induced by cold air is mediated by tachykinin release from sensory nerves. *Am. J. Resp Crit Care Med.* 151:1011-1017, 1995.

- 25- Zhang JX and Wolf MB. Effects of cold on microvascular fluid movement in the cat limb. *J. Appl. Physiol.* 71:703-708, 1991.

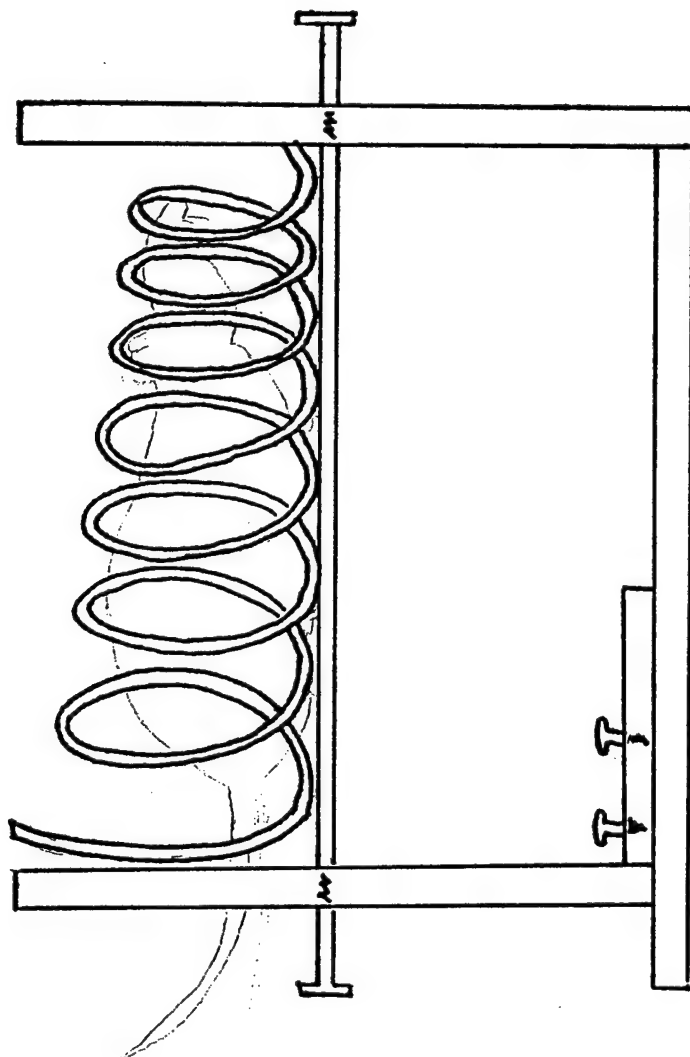
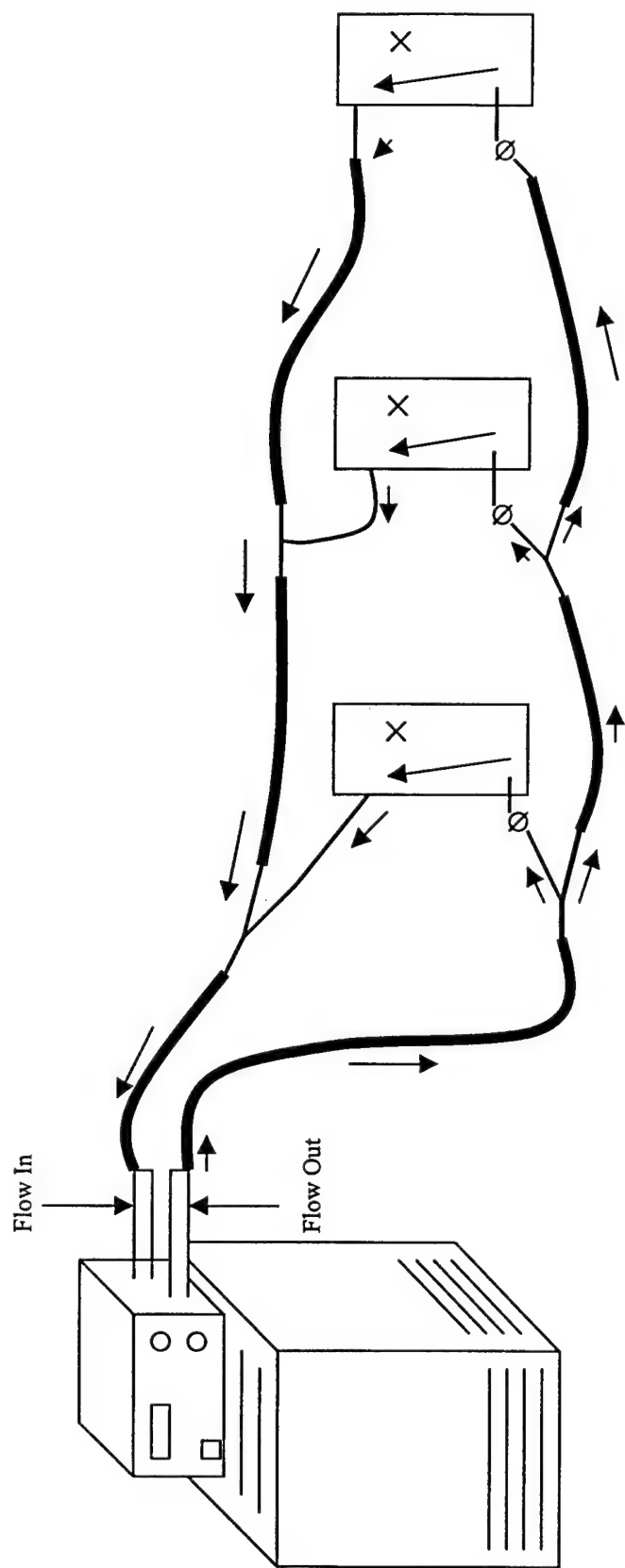


Fig. 1- The individual coils into which the rats were placed were constructed of $\frac{1}{4}$ " O.D. copper refrigeration tubing. The tubing was bent into a coil of 14 turns. The coil is supported on a platform made of plexiglass and stainless steel rods derived by removing rods from a previously described (16) restraining cage.



X- Coiled copper tubing through which water circulates.
 Ø - Stopcock for adjusting waterflow.

Fig. 2- This is a schematic of the 3 rat cooling system. The 3 coils are connected to a water bath in parallel using vinyl tubing, "Y" connectors, and hose clamps. A stopcock was placed at the inlet of each coil to regulate flow to each coil.

8-Oct-98

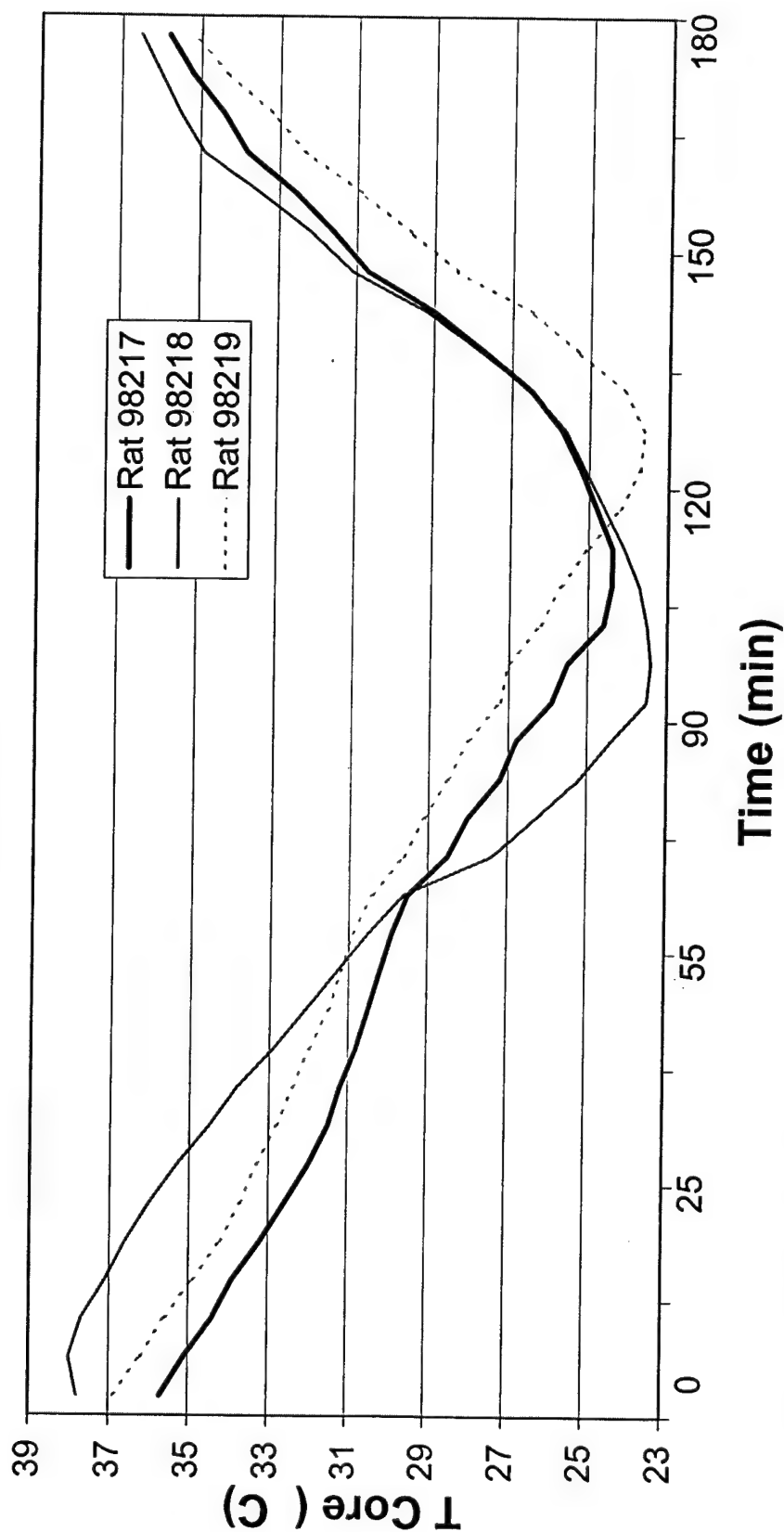


Fig. 3- Figures 3 represents the cooling curves of the 3 rats cooled simultaneously 8 Oct 1998. At time 0, T_{bath} was 15°C; over the next 15 min. the T_{bath} setpoint was moved to 5°C. At 45 min the T_{bath} setpoint was lowered to 3°C and T_{coil} stabilized at 5°C by 60 min. The lowest T_{core} attained was 23.4°C for 98218 at 100 min. At 120 min. the T_{bath} was increased to 25°C, at 130 min to 30°C, and at 150 min to 35°C.

20-oct-98

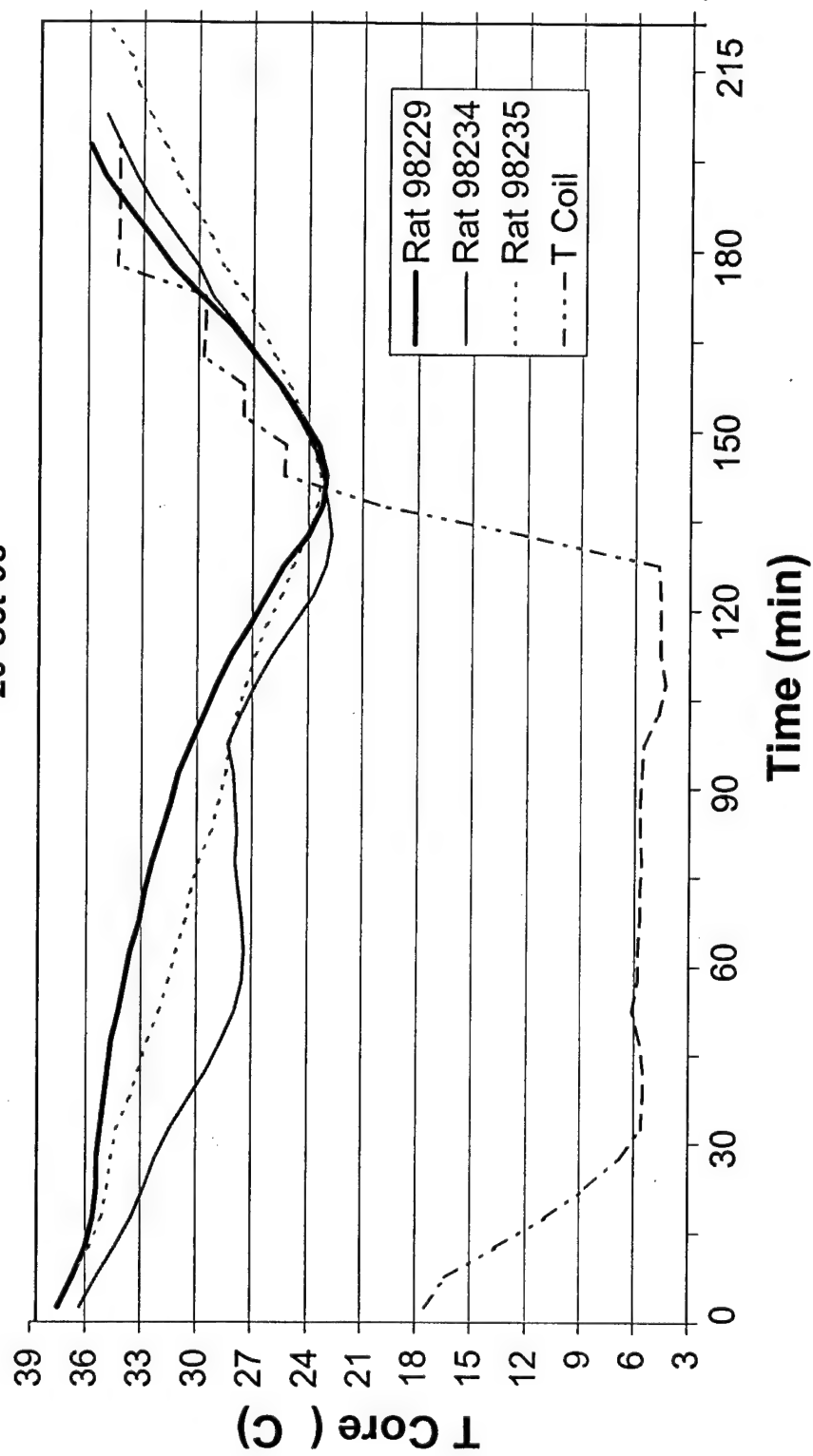


Fig. 4- Figure 4 represents the Tcore of 3 animals as well as the Tcoil for the 20 Oct 98 trial. The Tbath was decreased from 15°C to 3°C over the first 20 min. with the water entering the coil stabilizing at 5°C at 30 min. At 120 min., flow was stopped in all coils to maintain the Tcore's around 23°C for 30 min after which the Tbath was raised to 25°C, the flow restarted, and then Tbath stepped up to 35°C.

23-Oct-98

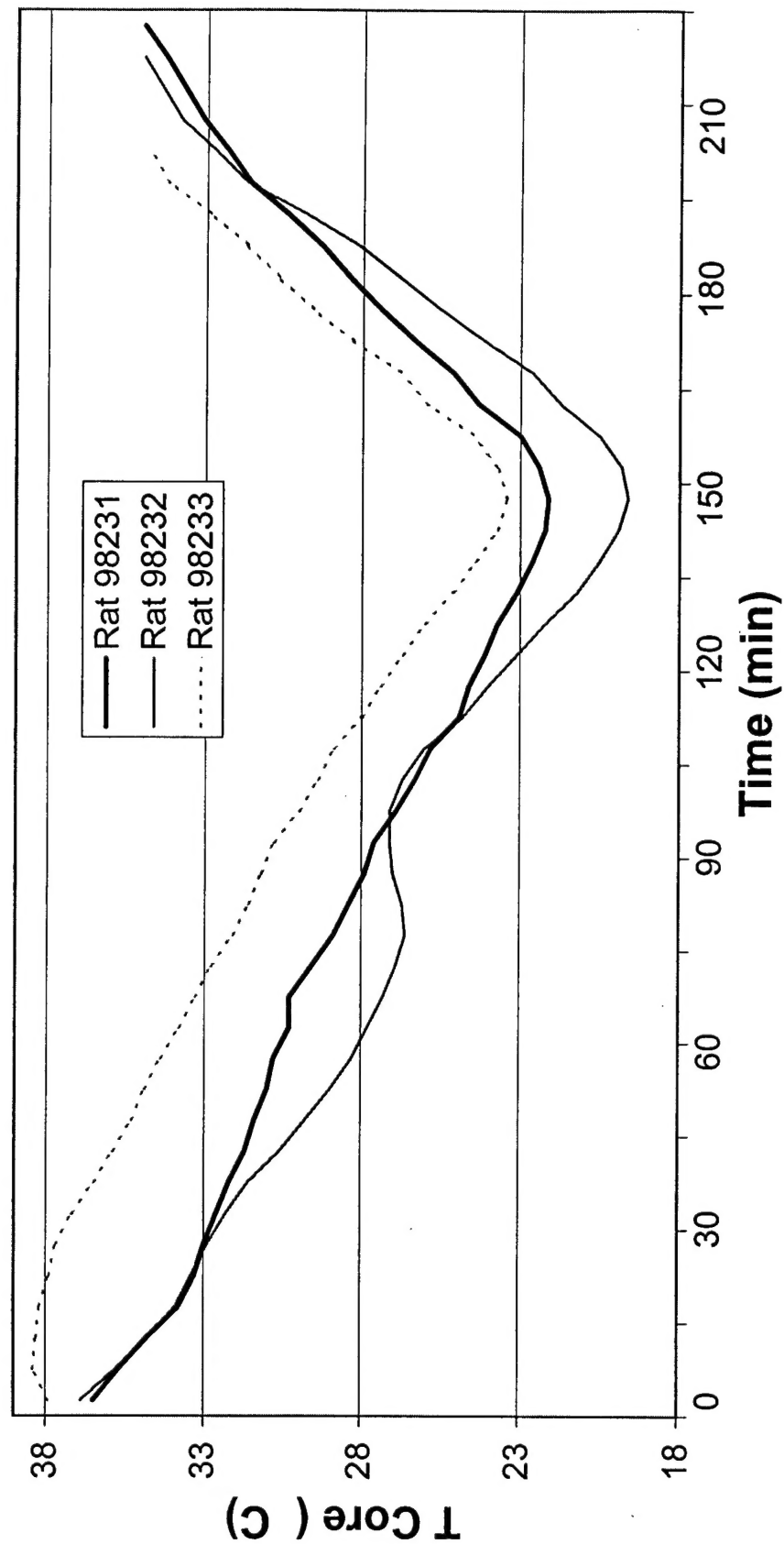


Fig. 5- Figure 5 represents the Tcore of the 3 animals cooled on 23 Oct 1998. Tbath was again decreased from 15°C to 3°C over the first 20 min. All animals were kept closer together than in previous trials by earlier modulation of flow into the coils. When Tcore of 98232 began to fall faster at 120 min, he was allowed to fall to 20°C. Rewarming was as in Fig. 3 and 4.

DISTRIBUTION LIST

1 Copy:

Defense Technical Information Center
8725 John J. Kingman Road, Suite 0944
Fort Belvoir VA 22060-6218

Office of the Assistant Secretary of Defense (Health Affairs)
ATTN: Medical Readiness, Pentagon
Washington DC 20301-0103

Commander
U.S. Army Medical Research and Materiel Command
ATTN: MCMR-OP
504 Scott Street
Fort Detrick MD 21702-5012

Commander
U.S. Army Medical Research and Materiel Command
ATTN: MCMR-PLC
504 Scott Street
Fort Detrick MD 21702-5012

Commandant
Army Medical Department Center and School
ATTN: HSMC-FR, Bldg. 2840
Fort Sam Houston TX 78236-6100

HQDA, Office of the Surgeon General
ATTN: SGPS-PSP, Preventive Medicine Consultant
5109 Leesburg Pike
Falls Church VA 22041-3258

Uniformed Services University of the Health Sciences
ATTN: Dean, School of Medicine
4301 Jones Bridge Road
Bethesda MD 20814-4799

Commandant
Army Medical Department Center & Schools
ATTN: Chief Librarian Stimson Library
Bldg 2840, Room 106
Fort Sam Houston TX 78236-6100

Commander
U.S. Army Medical Research Institute of Chemical Defense
ATTN: MCMR-UVZ
Aberdeen Proving Ground MD 21010-5425

Commander
U.S. Army Medical Material Development Activity
ATTN: MCMR-UMZ
504 Scott Street
Fort Detrick MD 21702-5009

Commander U.S. Army Natick Research, Development & Engineering Center
ATTN: SSCNC-T
Natick MA 01760-5000

Commander U.S. Army Natick Research, Development & Engineering Center
ATTN: SSCNC-S-IMI
Natick MA 01760-5040

Commander U.S. Army Soldier Systems Command
ATTN: AMSSC-CG
Natick MA 01760-5000

DISCLAIMER

The views, opinions, and/or findings contained in this report are those of the author and should not be construed as official Department of the Army position, policy or decision, unless so designated by other official documentation. In conducting the research described in this report, the investigators adhered to the "Guide for the Care and Use of Laboratory Animals, Department of Health and Human Services, revised 1996. United States Army Research Institute of Environmental Medicine is an AAALAC accredited facility and will continue to adhere to the standards and requirements thereof. Citations of commercial organizations and trade names do not constitute an official Department of the Army endorsement or approval of the products or services of these organizations.